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# DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

# Reconditioning the Neurosurgical Patient

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*San Francisco, California*

A new field has been opened for application of the principles of scientific medicine through the experimental approach and accomplishments of the reconditioning program undertaken during wartime by the armed services. This endeavor has marked the first organized attempt to establish procedures for the management of convalescence, up to this time a neglected phase in the natural history of disease. Nowhere can this be realized better than in the handling of injuries to the nervous system, which frequently are associated with prolonged periods of convalescence.

The general objective of reconditioning activities may be summarized briefly: to return the patient to normal life as soon as possible, in the best possible physical and mental condition. The methods used to accomplish this end in the armed services have been described in detail elsewhere.[1,2] The purpose of this discussion is to relate some of my experiences with neurosurgical patients, in order to stimulate others to think along the lines of convalescence management.

A scientific approach to convalescence must include physical and psychological factors. There is a growing body of physiological evidence concerning the significance of rest in the handling of disease, its uses and abuses.[3] Likewise, there is a slowly growing body of material seeking to establish simple objective tests by which a patient's capacity to perform physical work during convalescence can be evaluated.[4] Measurement of progress in convalescence is difficult because of the many factors operating upon the patient which usually cannot be separated. However, those physicians who have been associated with a well-functioning reconditioning service have been convinced that patients receive benefit from the activities, and these impressions are being substantiated by such studies as are in progress.

During the period of acute disease or injury, the physician often tends to lose sight of the patient as an integrated whole. This is reflected unconsciously in the way physicians often refer to patients as "a pneumonia" or "a gall-bladder." It is further reflected in the loss of interest which most physicians feel when they are assured that the disease process or injury is under control, and the patient is on the road to recovery. It is common for a physician to order "physiotherapy" for a patient in the convalescent stage, without bothering to specify the type or amount desired. Often the latter determinations become the actual responsibility of sub-professional personnel.

Yet there is no time when the patient as an integral individual requires more careful attention than during convalescence. It is during this time, usually chronologically the greatest portion of time lost from work due to illness, that the patient must make major adjustments to his condition, especially if a permanent disability will result. It is also during this time that regression in skills and physical status of the patient, not focally affected by his disability, may be prevented, and others minimized.

An effective way of presenting the idea of

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reconditioning is to refer to cases of transverse myelitis. Whether incurred by traumatism or by disease, there are few conditions which pose greater problems in care, morale, and adjustment. Prior to the war, transverse myelitis was a relatively rare condition, and perhaps because of this, not much thought had been given to the problems of adjustment, physical and mental, faced by the patient. It is not my intention to discuss the definitive treatment or prognosis of transverse myelitis. Others far more competent than I have already done so.[5,6,7] But I did have occasion to supervise introduction of reconditioning activities upon a ward service with a constant census of from 50 to 80 transverse myelitis patients. Most of them were received from 2 to 6 months after onset of the condition. They were beset in varying degrees with the associated curses: paralysis of the extremities, extensive decubitus ulcers, repeated urinary infections, urinary and fecal incontinence. The mere definitive medical and surgical problems were almost insurmountable. However, through excellent teamwork, lavish use of personnel and equipment, and with the help of new antibiotic agents, remarkable progress was made in organizing the definitive medical care that these patients received. Without these accomplishments reconditioning activities would have been impossible.

At the outset the patients were generally depressed. Though they realized efforts were being made in their behalf, they were not impressed. Many were in constant pain, requiring sedation, from the extensive sacral decubiti, in some cases extending down the thighs, in one case exposing both femora. Their approach to any new idea was negativistic, or at best passive. We planned a program as we might have planned an attack. One prime factor was the recognition that each man's status constituted an individual problem, and that as we obtained results with one, others would become encouraged and interested.

The first approach came through physical reconditioning activities. It was necessary to develop the shoulder girdle and upper extremities in these patients, in order to compensate for loss of function in the legs, which many of them would find permanent to greater or lesser degree. In this regard, the prognosis depends upon the level of the lesion, the nature of the wound, and the degree of spasticity. Individual evaluation must be made in each instance, and exercises must be directed towards development of strength in muscles which can assume compensatory function. In addition to the upper extremities, development of the abdominal musculature is particularly important, as these, in conjunction with the iliopsoas and quadratus lumborum muscles, may permit use of the locked knee braces, making it possible for the patient to ambulate without assistance. Elkins has recently presented an excellent discussion of such an evaluation as applied to an individual case.[8]

Trained physical instructors visited all the wards of the hospital to conduct exercises for bed patients. We assigned one instructor solely to the transverse myelitis ward. He spent the full day conducting group calisthenics and exercise involving the upper extremities, as well as individualized exercises for the lower extremities, depending upon the amount of function present. Swinging cross bars, exercise springs, and pulleys were installed on the beds, and patients were encouraged to use the apparatus as often as possible. In addition to regular exercises, patients used the swinging bars for moving around, making it possible for the patients to do many things for themselves. Similar aids allowing use of the arms and shoulders were installed. Thus, for example, a sturdy rope was suspended from the ceiling over the bath tubs, where patients were given daily treatments for decubitus ulcers. The patient was then able to swing himself into the bath from wheel chair or litter.

Games were organized and adapted. For example, a basketball could be tossed from bed to bed. The grass area adjacent to the ward was utilized in good weather. A large mat was placed there and a volley-ball net was erected. Patients were moved out, if their condition permitted, and played sitting volleyball from wheel chairs or sitting on the mats.

These activities were used in addition to the usual physical therapy methods, which were administered by physical therapists assigned to the wards. Within a short time muscular development improved in these pa-

tients. One patient who had been supine for several months was able to support himself on crutches with the aid of locked knee braces within two weeks. He had been unable to sit unaided on admission. As patients became able to do so, they entered a coordinated walking program. The first steps were taken in locked knee braces with crutches. The patient was given confidence by being suspended from the ceiling by means of a body jacket attached to a pulley which rode on tracks laid on the ceiling. In this way the patient was able to start his walking instruction without fear of falling. Later he graduated from the jacket to a walking frame, and ultimately, to the use of canes.

Watching these results had a tremendous effect on the morale of the ward. Every patient who succeeded in graduating from one stage of walking to the next acted as a stimulus to the others. The reconditioning activities made possible by efficient definitive treatment, in turn had a reciprocal effect. Relief of pressure, associated with suspensory exercises from the swinging bar, with more frequent changes of position, or with the use of crutches, resulted in more rapid healing of decubitus ulcers.

The second approach was made through organized recreation. Special Red Cross workers were assigned to this ward. Priority was given for all entertainment visiting the hospital. Motion pictures were shown at frequent intervals. Entertainment troupes and name entertainers visiting the hospital were always steered to the transverse myelitis ward before or after the performance in the hospital auditorium. Local groups were encouraged to visit the ward and to provide parties and game sessions as often as possible. Excursions were organized and on several occasions 20 to 40 of the more advanced patients were helped into a bus and taken to the country for picnics. Occupational therapists were assigned to the ward and made regular rounds with well equipped carts. The men were encouraged to participate in some form of craft work, both for amusement and for potential pre-vocational experience. Every effort was made to schedule activity for every hour, so that the patient would have no idle time to lie in bed pitying himself or brooding on his future. As a matter of fact, we probably overdid it in our enthusiasm, as we occasionally received a complaint from the patient that we were not even leaving him time to write a letter home.

Another approach was made through guidance activities. Every patient was interviewed shortly after admission. Information was obtained concerning personal problems (marital, pay, family, illness, etc.), and every possible effort was made to solve them, so that the patient was as free from external worries as possible. Other facts elicited included educational background, occupational interests and experience. This material was made the basis for evaluation of the patient's potentialities, and translated into occupational advice and guidance. Interviewers visited the patients at intervals during their stay, answering questions and searching out new problems.

The last approach came through educational opportunities and pre-vocational exploration. Actual academic instruction was offered in the form of small ward classes, where sufficient interest existed in the same subject, or through individualized instruction, using materials obtained through the United States Armed Forces Institute. A small electrical and radio shop was established on the ward, manned by a competent part-time instructor.

When possible, patients were moved to the various shops used in the general hospital program. These shops provided such opportunities as wood and metal work, jewelry making and lapidary work, radio repair, printing, photography and other graphic arts. Patient training and talent was used in conducting courses for other patients. Thus, one patient who had been engaged in radio work conducted a radio "workshop" which wrote and produced short radio programs on patient life. Through arrangement with a local radio station, these were actually broadcast and proved a stimulus to participation in the work.

One of the major automobile manufacturers made available an especially equipped car which could be operated by hand alone, and patients were taught to drive without use of their legs.

The net result of this program was an obvious and enormous lift in the morale of the patients. With an increased desire to recover

and with stimuli provided, showing the degree of recovery possible with all degrees of disability, the patients made a greater effort to conquer their handicaps. There was a steady decline in the percentage of patients who were bedridden, attributable not only to the recovery of neurologic function, but also to the optimistic tone introduced by attendents and the general revival of interests.

Many obstacles were encountered and overcome in the course of developing this program. As a new approach to a medical problem, there was resistance from both the administrative and medical staff. Simple medical care for these patients required much time and attention of nurses, orderlies and physicians. Medical personnel have been accustomed to few outside interfering factors on the wards. Thus, the physician prefers to make ward rounds as and when he finds it convenient. Conflicts between the need for holding classes and the need for seeing that each patient is allowed to soak in a tub bath had to be resolved. Many of these difficulties were disposed of through careful scheduling of activities, while success in conquering others depends upon the presence of adequate trained personnel and indoctrination in the objectives and reasons for the desired activities.

Patients with similar medical problems and degree of disability should be grouped together, thus permitting similar activities. This grouping also permitted introduction of simple modes of competition among patients who could compete on a fair basis. Every effort was made to utilize man's inherent competitiveness and desire for social approval, for example, by giving prizes for the number of times a man can chin himself on the bar placed on his bed, or for progress in study. Patients will compete to see who will be the first to graduate from wheel chair to walking with braces. Such simple contests introduce motivation effectively into the activities and increase patient cooperation.

While the program for transverse myelitis patients shows most dramatically what an organized approach to convalescence can do, the same basic facts hold for all neurosurgical patients. Nerve regeneration is a long, slow process and much time must be spent, encumbered by mechanical devices, to prevent contractures and ankylosis. Many patients are left with residual defects of function and others face the necessity of learning to use new neural pathways after nerve transplantation. The patient with peripheral nerve injury faces the same problems of convalescence and adjustment as the transverse myelitis patient, in less acute and depressing form and in many respects the same type of program can be applied.

Let us consider the case of a patient with peroneal palsy, secondary to an injury just below the knee which severed the nerve. For a time after neurorrhaphy the patient is immobilized in plaster. As a bed patient, he still has many needs to be satisfied. It is good medical sense to prevent regression of his skills and the physical status of his body as a whole. Beginning very shortly after operation, a trained instructor, supervised by a physician, can initiate regular periods of adapted exercises, helping to maintain the patient's general physical condition. The process of bedside guidance and education can be followed just as before.

After a period of time, the patient becomes ambulatory, at first in a cast and then on crutches. Immediately he can expand his scope of activities and visit the guidance department for more complete testing and orientation concerning his future. He may be tested for aptitudes and abilities and should find available information concerning the requirements of new occupations, in terms of physical capabilities, in the event that his disability will prevent return to his former occupation. He can visit the hospital prevocational shops and he can visit the gymnasium and participate in modified games and corrective exercises.

It was our custom to require each patient to spend a minimum of one hour daily on supervised educational and physical activities, in addition to such time as might be prescribed for physical and occupational therapy. As a patient progresses, he becomes more free to ambulate about the hospital, perhaps using a cane and foot-brace to counteract foot drop and the equinovarus tendency. During this period his physical activities may be broadened, he may use the athletic facilities under supervision and may be given supervised exercises in the swimming pool.

During this last stage of convalescence, which may be protracted, extensive and valuable use may be made of occupational therapy techniques. Thus the patient with peroneal palsy may operate a loom, a potter's wheel, or a jig-saw in spite of his injured leg. In such devices, weights and balances permit the machines to operate with graded degrees of effort, so that, as the patient's muscular control and strength return, effort required may be increased to produce fatigue.

Actually, in these cases, occupational therapy and physical reconditioning accomplish much the same ends as conventional physical therapy and corrective exercises. There is this difference: elements of competition and accomplishment are introduced, providing motivation. This keeps the patient at the exercise diligently over longer and more frequent periods. Interest is maintained when the patient is playing a game, or making some object to send home, such as a wallet. This is not likely to be the case when he is merely performing exercise "by the numbers."

All the processes mentioned aid in adjusting the individual to his situation, whether it is merely loss of time, or permanent disability. Limitations imposed by disabilities involve internal psychological readjustments as well as social and economic readjustments. The patient must adjust his reaction to a new situation. He must adjust to the reactions of others toward him and to his economic potentialities. A physician who loses the fourth and fifth fingers of his left hand can practice his profession with little difficulty. The same situation confronting a violinist is a major disaster necessitating a new occupation. To a greater or lesser degree such problems must be faced by all newly handicapped patients. The guidance afforded by a reconditioning program and its opportunities can ease the transition and adjustment to no small degree.

Our experience was obtained in a hospital which was well equipped and could afford an extensive reconditioning program. Ideally such a program requires a large hospital population, so that it is economically possible to invest in highly specialized equipment. For this reason it seems logical to advocate a trend toward the establishment in civilian hospitals of specialized treatment centers for related conditions. However, there are many valuable reconditioning measures which can be initiated in small hospitals, with a minimum of equipment and expense.[9] The most essential factors are imagination and initiative on the part of those responsible for the program and a coordinated drive to make the staff physicians aware of the patient as an entity during his convalescence. The slogan *"Your job isn't done 'till your patient's on the job"* should be popularized in the profession. When the physician recognizes his responsibility to provide good care during convalescence, means and facilities will be found.

*Summary*

1. A brief description is given of the objectives of reconditioning.

2. Application of reconditioning methods to neurosurgical patients is described.

3. It is emphasized that the physician's responsibility to his patient extends to the provision of adequate convalescence management.

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# Paranoia and Paranoiac Reaction Types

## (A Summary of Eight Case Histories)

CAPTAIN HAROLD ROSEN, M.C., and
LT. COL. HUGH E. KIENE, M.C.

In 1863 Kahlbaum for the first time used the term *paranoia* to designate the various persecutory and grandiose states. Thirty years later, Kraepelin[1] rigidly delimited our concept of the disease to that of a chronic and insidiously developing psychotic state with an exceedingly poor prognosis, endogenous in origin, without hallucinations but with the retention of clear and logical thought and action, and with all facets of the personality completely intact except for the presence of a persistent, enduring and unshakable, logically reasoned and closely knit delusional system.

Authorities quote various percentage ratios for the incidence of this disease, but in every instance the percentage is extremely low. Strecker and Ebaugh[2] saw only three cases of paranoia in over five thousand successive admissions (0.06%). During World War I, 69,394 neuropsychiatric casualties developed among U. S. officers and enlisted men (exclusive of native troops and troops serving in North Russia), but only 52 were diagnosed as "paranoia or paranoid condition". And during World War II, at least in the European Theatre of Operations, the disease seemed equally infrequent.

Between Jan. 1944 and April 1945, 14,000 patients were admitted to the 96th (U.S.) General Hospital (NP). Most of these were psychiatric rather than neurologic casualties. Paranoiac reactions were seen in only eight patients (0.06%). Because of the extreme rarity of the disease, the eight patients were studied in detail, and since all presented symptoms milder and apparently in an earlier stage of development than those which in civilian life usually force paranoiacs to submit to psychiatric care, an attempt was made to obtain information and draw conclusions about pre-military adjustment, psychotic and pre-psychotic personality traits, and possible etiological factors, in an endeavor to arrive at a better understanding of the underlying psychopathology of the disease process itself.

Five of our patients were nurses, one was a medical officer and two were line officers. One of the nurses was a captain and the medical officer was a lieutenant colonel, as was the ranking line officer who, so we had been told, had been recommended for promotion to a full colonelcy. In each case, systematized and logically presented delusions of reference, insusceptible to logic and with retrospective falsification of memory, were present. Each patient showed an irresistible selection of environmental evidence in an attempt to involve more and more individuals in the conspiracy against him. With one exception (patient F), delusions of grandeur were minimal. Rationalizations were plausible and the fundamental premises credible. Delusional systems were in an exceedingly early stage of development.

Patient B was a 36 year old nurse who believed fellow nurses jealous because of her undoubted efficiency. With her present knowledge, she now understood that this alone accounted for the innumerable slights, snubs and transfers that she had suffered during her thirty months of army service. The discriminatory attitude of her fellow nurses had naturally enough communicated itself to ward-men and patients, incited them to direct disobedience of her orders and even to actual insubordination, and was basically responsible for the disciplinary problems with which she was so frequently confronted, although it was only now, after so long a period of time, that she was able to realize this. Also, as she now saw, an added factor had soon inevitably come into existence. This unfairly gained reputation had constantly preceded her arrival at each new post and as a result medical officers, by expecting friction to develop between herself, fellow nurses, corps-men and patients, unconsciously helped create and intensify such disciplinary problems. Favoritism was therefore ultimately shown an inexcusably insubordinate corps-man at the last hospital to which she had been assigned,

and we at this hospital had finally joined the conspiracy against her by making a completely unjustifiable psychiatric diagnosis, thereby punishing her instead of the responsible and reprehensible culprit. Her delusions colored and distorted every aspect of her personality. Ever widening environmental factors were constantly being incorporated into their structure. She was therefore transferred to the Zone of the Interior, firmly convinced that she was being persecuted, but with all facets of her personality otherwise intact, a rigid, inflexible and uncompromising individual whose difficulties she attributed fundamentally to the fact that, because of her own efficiency, nurses and medical officers had conspired to have her sent back to the States, in order to protect an inefficient wardman from the military penalties of insubordination.

Patient D was a 41 year old chief nurse, efficient, rigid and socially inadaptable, who was unalterably and unshakably convinced that fellow nurses had conspired to have her railroaded out of the European Theatre of Operations perhaps, as she hinted, because her assistant wished to replace her as chief nurse. Her subordinates had naturally enough joined in the conspiracy and so had the psychiatrist. Retrospective falsification of memory was present.

Patient F was a line officer, a second lieutenant who was hospitalized for psychiatric observation because of the friction which arose within his command as a result of his overzealousness in executing orders, as well as because of his extreme condescension and superciliousness in his contacts with other human beings. Since the age of seven, his dominant idea had been to fly to the moon, and during the past two years he had, so it seems, finally perfected the necessary apparatus.

These presenting symptoms were typical of those shown by all eight patients. In the last analysis, however, the diagnosis of paranoia can be made only on the basis of the whole developmental history of the individual patient. For this reason, because of their length and the wealth of pertinent material obtained, the detailed case histories of these patients will be the subject of a separate paper. Each patient showed circumscribed delusions and nothing more. These invariably took their origin in actual fact and each additional fact uninterpreted helped further confirm the fundamental delusion. The patients themselves were impervious to reason. Their delusions, on close examination, seemed out of harmony with their educational background and environmental surroundings, served as a unifying thread whereby the experiences of a life-time became integrated one with the other, and seemed to constitute an integral component of the personality. All eight may be considered as rigid and unyielding paranoiac personalities who, in the still more rigid and unyielding environment of the military situation, resorted to systematized but circumscribed paranoiac symptom formation.

All eight were exceedingly intelligent. Patient D held the degree of master of science in nursing arts and for years had taught the subject in various first-class nursing schools. For three years, patient G was the proud possessor of a coveted surgical fellowship; in addition, he later became a member of the surgical faculty of a grade-A medical school. Patients A, B and G attained scores of 116, 124 and 128 respectively on the Wechsler-Bellevue* scale. In every respect, therefore, aside from their inability to achieve a satisfactory adjustment with their fellows in the environment in which they found themselves, these patients seemed possessed of superior intelligence.

### *The Personality Picture*

Kraepelin[4] comments upon a psychopathic disposition, present in most of his patients in the form of irritable excitement with occasional rough and violent behavior, manifested by distrust, obstinacy and a sense of fore-ordained birth to high and lofty achievement. Kretschmer[5] postulates a characterological basis in a refined, hypersensitive, seclusive, shy and easily hurt individual with deep emotional feelings, high ethical standards and broad cultural interests, and considers such individuals a special reaction type in whom the disease is precipitated during the course of specific life experiences. Heil-

*On the Wechsler-Bellevue scale, an I.Q. of 91 to 110 is considered "average"; 111 to 119, "bright normal"; 120 to 127, "superior"; and 128 or over, "very superior".

bronner[6] theorizes the presence of a "characterogen"; paranoid symptom-formation, so he believes, may be produced as a result of mental exhaustion coupled with traumatic life experiences in the hypersensitive and brooding, highly ambitious, conscientious and meticulous, good-natured, well educated and highly cultured type.

Our patients, superficially at least, showed all these traits. In addition, from childhood on and to a marked degree, they seem to have shown a combination of all the personality traits that Jones[7] considers characteristic of the god complex, that Hendricks[8] attributes to the narcissistic character, that Miller[9] finds indicative of a poor prognosis in paranoid disorders, that Cameron[10] developes in his consideration of the origin of paranoid thinking, and that Adolf Meyer[11] and Montassut[12] describe as the character pattern of the paranoiac. These traits are summarized by Brickner[13] in his descriptive phenomenological account of the paranoid character, as the reaction pattern of sulky, disagreeable and arrogant, self-important and suspicious individuals who long to dominate, blame others freely, quickly sense insult, and believe others wish them injury, considering themselves thereby victims of persecution.

Our patients not only manifested these traits in toto, but showed marked obsessional-compulsive personality trends as well. None of these arose de novo; all were present during childhood. As children, at least six had apparently been aloof, suspicious and seclusive, stubborn, obstinate and resentful of discipline. Three at least (B. C and D), and most probably all eight, had shown marked temper tantrums and pronounced compulsive traits. When crossed, they became sullen, morose, sulky, peevish and irritable. Not one, so far as we could determine, had for any considerable length of time been able to remain at congenial play with other children. As a result, not one could be said to have had a normal childhood.

As adults, their personality picture remained unchanged. All eight were conceited, egotistic and egocentric. Their memories, so they claimed, were infallible. They never were and never conceivably could be in the wrong. They were incapable of mistakes. And this exalted self opinion was constantly evidenced in their excessive self-confidence, their proud and head-strong attitudes, and their constant hypercritical belittling of the efforts of others. Patients B and E admitted matter-of-factly that they were professionally and socially superior to their associates. Patient D, despite her lack of psychiatric training, all but attempted, within a few days of her transfer to duty in the neuropsychiatric section of a station hospital, to introduce thorough-going reforms in an effort apparently to obtain administrative control of the section. Case C, to quote acquaintances, associates and patients, "acted like a little tin god". And D and G were castigated by indignant exclamations like "Who do they think they are? God Almighty?"

All eight showed limited capacity for identification with their fellows. They were completely insensitive to the needs of others. Most were epitomized as "good at bossing". All appeared asocial, aloof, inaccessible. No real transference was ever achieved. They were verbose, sententious, circumlocutious and grandiloquent. Their command of English at times seemed as exact and precise as their own meticulous personalities. Nevertheless, they at no time made spontaneous or revealing statements about themselves. Seemingly frank answers on their part served actually to conceal their thoughts, to maintain and even increase their characteristic inaccessibility.

They could be pleasant, good-natured, charming and even witty, provided only that they had their own way. All eight were keen, cultured, highly educated, with comparatively broad interests. But they manifested a need for praise so acute as to force them to act always as though hide-bound by tradition. They were hypersensitive to the mildest of criticism. They could accept no suggestion, let alone the discipline imposed by the army on even its highest ranking personnel. They sulked, remained morose and sullen, sat apart and refused at times even to eat or talk with fellow patients (cases B, C and E). Or they became irritable and peevish, developed marked impatience, engaged in temper tantrums, and showed themselves stubborn and obstinate, hard-headed and head-strong, argumentative, arrogant, dictatorial and domineering (cases A, B, D and H).

They were socially inflexible and unadaptable, uncompromising, incapable of making even the most minor of concessions. In their work, they were rigid and over-zealous, meticulous, over-conscientious, inelastic. They insisted on the immediate, exact, precise and detailed execution of regulations as they alone interpreted them. They showed that inward rigidity of thought and loss of flexibility so characteristic of the paranoid and the paranoiac.

All eight were ambitious and aggressive, and difficult to get along with. All eight found it imperative to force others to conform to their morals, their work and their ideas. Patient D refused to allow her subordinates to drink cocktails, and G enjoined his personnel from sitting on their bunks. Six were hospitalized because their efficiency, over-meticulous beyond the exercise of common sense, had created sufficiently pronounced friction and discontent within their commands to constitute a definite morale problem.

All eight, despite their marked projection tendencies, rationalized so plausibly and with such inexorable logic on premises so superficially credible as to deceive at first even the disposition boards which examined them. Fault-finding is common in the army. Complaints of individual injustice are rife. The letting off of steam in this way is even encouraged; both enlisted men and officers indulge in it constantly. And adequate reports of the immediate pre-hospitalization difficulties of these patients were seldom available. As a result, even trained psychiatrists could at first be hood-winked by their rationalizations that the cards were stacked against them because of their insistence on functioning in what they termed "not the army way ... but the right way". The severe distortion of their personalities could be seen only after prolonged observation.

### *Possible Predisposing and Etiological Factors*

An attempt was made with these patients to unearth the various factors that at one time or another have been postulated as basic for the production of paranoia. Heredity has frequently been indicted. So have race, constitutional type, sex, age, life period, brain pathology and endocrine dysfunction. Feelings of inferiority are frequently present, and on occasion their roots have been traced to emotional insecurity during infancy and childhood, poor social and economic adjustment, or frustration as the result of continued failure to achieve subjectively overevaluated goals. Pronounced guilt feelings, auto-, hetero- or homo-sexual in origin, may often be unearthed, usually in association with definite sexual maladjustment or actual homosexuality. And various authorities postulate either a constitutional predisposition or a special reaction type in which some, but not all, believe the symptomatology is precipitated as a result of specific traumatic life experience.

*Heredity:* Only one of our cases showed what some investigators could perhaps consider a hereditary taint. Patient H had three "nervous cousins," and a second cousin who had been committed to a mental institution. But the possibility of a taint existed also in the case of patient C, since her father had become a chronic alcoholic while she was still a child. Nevertheless, the mentally sound and the psychiatrically ill show no significant difference in the number of alcoholic ancestors who preceded them.[14] Since at least six of our eight patients showed no abnormal hereditary background, we therefore do not believe that inherited and inheritable factors are of any significant predisposing or etiological importance so far as this particular series of cases is concerned.

*Body Build:* According to Kretschmer, paranoiacs are constitutionally asthenic. Among our patients, cases A, C and G were typical leptosomes, but E was pycnic, and B, D and H pycnoid. As a result, these patients show insufficient uniformity of body type to be considered confirmatory of Kretschmer's findings.

*Sex:* Most authorities believe paranoia to be predominantly a disease of the male.[2,15] Nevertheless, five of our eight patients were nurses. This poses the problem of why more than half our cases should have occurred among women. Nurses receive direct commissions in the Army, and are therefore better able to continue along their own paranoiac way, as detailed in the case histories of the individual patients, until the demoralization

of their subordinates has either resulted in an investigation of their activities or reached the point of requiring their hospitalization for psychiatric observation. Enlisted men and members of the WAC, on the other hand, for the most part never receive promotions enough to make them a comparable focus of demoralization before being transferred from post to post, diagnosed perhaps as paranoid psychopaths, and discharged from the army by one means or another. Furthermore, since such patients impress non-psychiatrists as being mentally well, line officers, enlisted personnel, medical officers serving with line outfits and perhaps even physicians in fixed medical installations would most probably be handled through administrative channels. But we tend to be protective of women, and those who serve as nurses in fixed hospital installations would therefore, so we believe, be more apt to be disposed of by medical rather than administrative channels.

*Age:* With two-thirds of Kraepelin's patients, the psychosis had its onset after they reached the age of thirty. Most were between thirty and forty. Of our patients, one was 33, two 36, one 41, one 46, one 50 and one 52. This seems to indicate that paranoiac symptomatology may produce social maladaptation (and therefore manifest itself in what for the most part is considered the onset of the disease), at somewhat higher age levels than those usually postulated for this psychosis. Nevertheless, a definite distinction must be made between actual and apparent date of onset. The patient, for the most part, reaches the psychiatrist only after social adaptation has become obviously impossible. Paranoiac symptoms develop gradually and insidiously over a long period of time, the exact length of which in most cases it seems difficult if not impossible to ascertain because of the retrospective falsification of memory so characteristic of the disease. Over half of Kraepelin's patients, for example had lived in freedom for nine years or more.[4]

Patient E (aged 46) had developed litigious behavior severe enough to warrant psychiatric observation while still a civilian. Hers was obviously a case that had been in the process of development for perhaps a decade. Patient G (aged 52) had been under psychiatric observation at least a year before his behavior in the European Theatre of Operations warranted boarding him to the Zone of the Interior. For patient D (aged 36) we hesitate to hazard any guess whatever about the length of time it had taken symptoms to develop. Patients C and D (aged 31 and 36 respectively) were perhaps early cases, at least so far as the development of the delusional system itself is concerned. And patient A (aged 33) seemed to be at the earliest possible stage of delusional systematization. At least five of our patients therefore seem to have been between thirty and forty at the time of the actual onset of the disease.

*Life Period:* So high an age grouping brings to the fore the possibility of the climacteric as a precipitating factor. Our patients, however, all denied either increase or decrease of sexual desire. The five nurses admitted to no menstrual irregularity, amenorrhea or other menopausal symptoms, although patients E and C (aged 46 and 41 respectively) were constantly complaining of sexual immorality or laxity on the part of their associates, a delusion ultimately and questionably perhaps due to decrease of libido on their part. Patient E, so we believe, had most probably been litigious and paranoiac for a decade or more. In addition, it seems worth stressing the fact that the personality picture of these patients, so far as we could determine, had shown no significant change during the two or three decades preceding their hospitalizations. As a result, neither the endocrine factor nor the physical or psychic stress of the involutional period are believed to have exerted any significant etiological effect in precipitating symptoms in these two patients.

*Other Organic Conditions:* At no time had any of these patients suffered head trauma. Their teeth were in excellent condition. No medical evidence was found indicative of foci of infection, although two (A and D) had previously had cholecystectomies. They drank but sparingly, took no drugs, and were serologically negative. In every respect, so far as we could determine, their physical condition was excellent.

*Interaction of Individual and Environment:* Our patients were all anal-sadistic individuals whose psychoses seem to have been pre-

cipitated by sudden promotions to positions of authority. According to Krafft-Ebing, the psychosis results from the interaction of individuals, defectively constituted in the self-regarding and social instincts, with a world which refuses to accept them at their own self-evaluation. To some extent at least, this seems true for each of our cases. Patient G, for instance, was an excellent surgeon. The more meticulous and precise his operative technique became, the greater the surgical ability to which he attained. Within office and classroom, he could be as rigid and domineering as he wished. But once he was assigned as commanding officer of an army hospital unit and attempted to force the individual members of his command under the rigid, hair-splitting and detailed minutiae of his orders, his aspirations for the domination of his subordinates were soon checked by their inability to observe his demands, the friction that consequently developed, and their definite complaints that ultimately resulted in his hospitalization. And such frustration, for him as well as for patients B, C and D, may actually have been the factor which ultimately precipitated the appearance of delusional trends.

*Inferiority Feelings:* Most authorities stress the fundamental importance, in the psychogenesis of paranoia, of feelings of guilt and inferiority. With all our patients, feelings of inferiority seem well masked by the megalomania that characterized them. Clues in profusion, however, were to be found in their pathetic craving for praise and recognition, their continued dependence on the good opinion of their associates, and their hypersensitivity to the slightest whiff of adverse criticism. They constantly betrayed their underlying insecurity and sense of inferiority by their exact and detailed observance of each and every social custom, by their psychaesthenic over-zealousness in the performance of the minutiae of their individual duties, and by the habitual hypercriticism with which they so constantly belittled individuals and ideas other than those they themselves patronized and promulgated.

Such feelings of inferiority most probably have multiple roots. Patients C and E had both suffered financial insecurity early in life, and so most likely had patients D and G. All eight had over-evaluated their aims in life, were intensely ambitious, and had shown marked aggressive drives. As a result, because of their obsessive-compulsive make-up and their perhaps consequent incapability of associating on congenial terms with their fellows, they had suffered inevitable frustration from their inability to achieve the goals set by their inflated ambitions.

But over and above all this, during childhood emotional insecurity had been pronounced. When directly questioned, all eight patients claimed that their younger years had been especially happy ones. From outside sources, however, it was learned that patient C had on numerous occasions characterized her childhood as the most miserable period of her life, and it soon became apparent that the halcyon statements of the others as well were the result of pure rationalizations on their part. Six had lost at least one of their parents during childhood. The mother of patient H had died during childbirth. Patient B had frequently been reprimanded by her father because of an apparent sadistic streak which manifested itself in her treatment of animals and other children. The father of case C was an impecunious alcoholic. Two patients (B and C) had received too little love from either parent; in fact, B seems to have been raised alternately by grandmother and aunt, and C was constantly in straitened financial circumstances, was separated from her siblings, and was raised successively by various neighbors and friends. And after their respective mothers died, patients D and E had felt the crushing weight of their self-imposed responsibility for raising their numerous siblings.

But feelings of inferiority may have other roots as well. Of these, probably the most fundamental are those which give rise to sexual conflict. So far as our patients are concerned, this can be discussed most simply under the twin rubrics of general sexual maladjustment and homosexuality.

*Sexual Maladjustment:* Sexual maladjustment of one type or other is to be seen in every case reported in full in the literature. All of Bleuler's carefully studied patients show what he characterizes as weak sexuality, due either to insufficient drive or to strong inhibitions.[15] At least six of our cases showed this

weak sexuality. All claimed what they termed a normal sexual and heterosexual development, but all nevertheless showed evidence of maladjustment in this particular constellation. Only two dared attempt the marriage relationship. Patient B chose as her husband a chronic alcoholic eleven years her senior, whom she divorced after four years of married life and with whom she seemed sexually incompatible, although from her hints it was impossible to learn whether this incompatibility was due on her part to actual or relative frigidity or on his to satyriasis or sexually perverted practices. The exceedingly short-lived marriage of patient H did not take place until he was 41. Cases D and E both avoided marriage because of the heavy responsibility they assumed due their respective families. D was unfortunately never able to meet the man she could wish to marry, and A, D and E had each vainly tried to enforce an exceedingly rigid moral code upon associates and subordinates. D especially felt an almost overpowering sense of frustration because of her inability to prevent the nurses under her command from associating, however innocently, with officers whom she knew to be married. E believed herself discriminated against because of her frequent and constant protesting about the alleged laxity of morals on the post to which she had been assigned. And finally, C apparently gave the impression of being a vain and coquettish exhibitionist who, despite the fact that she preened herself above all else on her sexual prowess, nevertheless considered intercourse an activity designed for her own especial delight, and therefore had apparently never passed beyond what in the male would be termed the phallic phase of her psychosexual development, if in point of fact she was not actually frigid.

*Repressed Homosexuality:* Homosexual conflicts are frequently present in paranoia.[2,14,16] Psychoanalysts believe that the basic mechanisms involved are those concerned in homosexual fixation, attempted suppression of the homosexuality with failure of sublimation, and subsequent repression with later return of the repressed material in distorted form.[17,18] At no time were we able to attain sufficient rapport with any of our eight patients to make possible a consistent probing for whatever homosexual elements might be present. Clues existed, but these may easily be overinterpreted. Early in life, five had lost the parent of the same sex as the patient in question. In each case, the prime persecutor was of the same sex as the patient persecuted: it was the medical major with whom he had had trouble who finally made it possible for higher authority to discriminate so openly against G; their fellow nurses were conspiring against patients B and C because of jealousy evoked as a result of their outstanding efficiency; D had been persecuted so that her assistant could seize her job; so had A's first chief nurse, with whom A identified herself; and E's chief nurse had deliberately stigmatized E with a psychiatric diagnosis in order to be free to continue indulging in sexual relations with the commanding officer of the post.

Their over-emphasis on the morals of fellow nurses can perhaps be interpreted as unconscious attempts on the part of patients D and E to retain and even protect potential love objects. The semi-erotomania of C, if true, can possibly be considered as overcompensatory, and the probable frigidity of B may also have had homosexual roots. In addition, these may have conditioned the exceedingly positive avoidance of marriage that characterized patients C, D, E and G, irrespective of the rationalizations they themselves advanced.

In view of the fact that, psychoanalytically speaking, constipation in patients with persecutory delusions is frequently conditioned by conflict about denied but nevertheless projected anal-sadistic tendencies,[19] it seems of parenthetical interest to note that not one of these patients at any time complained of constipation. The possibility of overemphasizing these factors should therefore be stressed. Nevertheless, we believe that a homosexual element is probably present, but because of our tenuous rapport with the patients in question, not enough material could be unearthed about their developmental histories or the psychodynamics of their individual cases to make it possible for us either to prove or to disprove this hypothesis.

### *Summary and Conclusions*

1. Of over 14,000 consecutive admissions

to the only general hospital in the European Theatre of Operations whose case load during World War II was continuously neuropsychiatric, only eight were diagnosed as cases of early paranoia. The composite personality picture of these patients has been drawn during the course of this article.

2. Individual psychotherapy was of little value, primarily because our paranoiac patients considered themselves psychiatrically well, identified the therapeutic attitude with the persecutory system, incorporated the psychiatrist in the delusional trend, and made it impossible for adequate rapport to be gained or maintained.

3. No organic or somatic factors were unearthed that could be considered as responsible for the appearance or development of the psychosis. The physical type varied. Neither life-period nor endocrine dysfunction seemed at fault. Nevertheless, the disease apparently had its onset, for the most part, between the ages of thirty and forty.

4. On the other hand, our patients all suffered emotional insecurity early in life, showed an unsatisfactory sexual adjustment, felt basically inferior, inadequate and insecure, and manifested a craving for recognition unattainable in the environment in which they found themselves, especially when one takes into consideration the fact that the military environment itself, under certain conditions, can be even more rigid and unyielding than their own exceedingly rigid and unyielding personalities.

5. As a result, we subscribe to the view that in these individuals, who for some reason or other were so predisposed, traumatic life experience initiated the insidious advance of paranoiac thinking, with its irresistable selection of environmental evidence, to the personality distortion of paranoia. In the present state of our knowledge, however, it seems impossible to make any statement more definite than this.

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# Neurological Clinical Pathological Conference

of the
Cincinnati General Hospital

CHARLES D. ARING, M.D., *Editor*

Case No. 192873

## *Presentation*

A 73 year old white man was brought to the Receiving Ward on the evening of April 23. He noted that his right scrotum had swelled and had become painful five days previously. He had not been particularly inconvenienced by what he said was a hernia for which he had worn a truss for one year; but felt that he should have it repaired since it was no longer reducible and was painful. There had been anorexia and flatulence since the scrotum had swelled. The patient had been having intermittent headache for about a month; they were more frequent after the scrotal swelling had become established.

A year previously he had been thoroughly examined in the Out-Patient Department. He had a productive cough after a cold. He told of a single episode of sudden unconsciousness two years before, calling it a "nervous breakdown". The only abnormal findings in the clinic examination were blood pressure 150/90; the electrocardiogram showed left axis deviation, slurred QRS complex in all leads, and an aberrant lead 4; in roentgen examinations there was a large amount of air in the stomach and osteoarthritis of the mid-dorsal vertebral column.

After an initial examination in the Receiving Ward on April 23 it was decided that he had a hydrocele and preparations were made to admit him to the Surgical Ward. When the patient was again seen a few moments later he was having a convulsion which was followed by semicoma. He aroused when disturbed; he was sweating profusely and did not use his left extremities voluntarily. There were Babinski signs bilaterally.

He became clearer on April 24 and cooperated for a thorough examination, falling asleep toward the end of it. The following abnormalities were observed: the right testicle was obscured by a large fluid accumulation which transmitted light. The palpebral fissure on the left was twice as wide as that on the right; the entire forehead was maintained constantly wrinkled. The pupils were 1.5 mm. in diameter and reacted well. He was unable to perform any eye movement besides downward gaze and convergence; the eyes spontaneously tended to cross. On attempting downward gaze the right eye remained depressed longer than the left, they both described coarse, vertical nystagmus. On attempting upward gaze the eyes diverged slightly and coarse vertical nystagmus resulted. On trying to look toward the right or left the eye furtherest from the object sank, then shortly returned to its original position, the other eye not moving. He did not fix the eyes on an object when the head was passively turned, even though he was requested to do so. He did not appear to appreciate pinprick in the distribution of the left fifth nerve. There was a left facial weakness including the ocular orbicularis. There was mucus in the throat and the speech was thick and barely intelligible. He swallowed without difficulty and the tongue protruded in the midline.

There was paralysis of the left upper extremity and moderate to marked weakness of the left lower. When he attempted to hold the right upper extremity outstretched it described large, oscillating movements. When the arm was tapped the involuntary movement became wilder, it continued unabated until the extremity was put to rest. Resistance to passive movement was reduced in all extremities much more so on the right. There was complete lack of checking in the right upper extremity. Ataxia and tremor were considerably less in the right lower than in the right upper extremity. When he sat up, the trunk weaved about and he leaned toward the right, the head turning toward the right and the chin sagging in the same direction.

It was impossible to evaluate sensation. Earlier he had not moved the left extremities on pricking them; he had removed the right

extremities from pinprick. Twenty-four hours later pinprick seemed to be appreciated in the upper extremities. The deep reflexes in the left extremities were more active than those on the right, the abdominal reflexes were unobtainable; there were extensor plantar responses bilaterally.

He had to be catheterized repeatedly. He had a temperature of 101° F (rectally) on admission and a pulse rate of 90. On April 25 his respirations became labored and his temperature rose abruptly to 108°, and he died after 50 hours in the hospital.

*Laboratory Data:* Blood counts and urinalyses were normal, and a blood Kahn was negative. The blood urea nitrogen was 19 mg.%, the non protein nitrogen 40 mg.%. A lumbar puncture done on the evening of admission revealed an initial pressure of 50 mm. of water; the fluid resembled water and contained 1 lymphocyte per ccm. The Pandy test was negative as was a Wassermann reaction. It is not known why a quantitative protein determination was not done.

### *Differential Diagnosis*

Dr. H. Houston Merritt:* I always console myself in a presentation such as this that it does not matter about the right diagnosis; of most importance is the elucidation of the symptoms and signs. Let us select some salient points to see if we can localize the disorder, and then try to determine the nature of the lesion responsible for the death of the patient. This man presented himself at the hospital because of a mass in the scrotum, which was determined to be a hydrocele, and he was admitted for treatment of it. I think there we shall leave that bit of the problem, as I do not feel the hydrocele had anything further to do with the terminal illness.

It is important that in the past history this man had an episode of unconsciousness which was called a nervous breakdown. Though the data are limited it is probable that he suffered a cerebrovascular accident, a small thrombotic lesion in a silent area of the brain. The basis was most likely arteriosclerosis, as the man was then 70 years old and had evidence of cardiovascular disease.

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We may now pass to the day when the patient was to be admitted to the hospital. The Admitting Physician evidently left him for a moment and when he returned the man was in semicoma following a convulsion. This convulsion is not described but we presume it was a grand mal seizure with tonic and clonic movements.

The patient aroused somewhat from coma and on the next day he was examined closely. There was inequality of the palpebral fissures, the left was greater than the right, suggesting either that the left fissure was wide due to facial weakness or the right narrowed due to sympathetic or third nerve involvement. The pupils were small and reacted well. Alteration in the size of the pupils may be caused by a number of lesions, the disturbance may be in the peripheral or in the central nervous system. The small pupils when taken together with other evidence would suggest that there was involvement of the descending sympathetic pathways. The eye movements were disorganized; he could look downward and he could converge and the eyes tended to cross spontaneously.

On attempting to look toward the right or left, the eye further from the object sank, indicating weakness of internal rectus on lateral deviation. There was weakness of the external rectus of either side. Disturbance of the posterior longitudinal bundle also seems indicated. The disorder was so bizarre that we can not say just which nuclei were hit.

The left facial weakness included lid movements; this may indicate either involvement of the seventh nerve nucleus or of the aberrant corticobulbar tract that sends fibers to the seventh nucleus. There was difficulty in the speech but swallowing was strong. This suggests involvement of the nucleus ambiguus or bilateral corticobulbar tract lesion, i.e. the swallowing difficulty was a symptom of pseudobulbar palsy.

Examination of the motor system revealed paralysis of the left side of the body greatest in the upper extremity. We find that in addition to paralysis on the left there was disordered movement of the right limbs, more marked in the arm than in the leg, of the type seen with lesions in cerebellar pathways. He had involuntary movements in the right arm when it was outstretched, large move-

ments which became wilder and wilder. An alternating flexion-extension type of movement is spoken of as "wing-beating," was it so described in the chart?

Dr. Charles D. Aring: No.

Dr. Merritt: Such movements are common in Wilson's disease and they are not uncommon in multiple sclerosis. It is my feeling that this type of tremor is seen with involvement of the superior cerebellar peduncle. He weaved and leaned toward the right, suggesting disorder of the vestibular apparatus.

We find that he didn't appreciate pinprick in the distribution of the left fifth nerve. Also he did not seem to appreciate pinprick with the left extremities, at least he did not remove them from it. The difficulty in interpreting this symptom is that he also had weakness of the muscles of the left limbs. The left-sided sensory involvement could be due to a lesion of the sensory tracts in the cortex or thalamus or high in the right brain stem above the point where the fibers from the face cross to join the fibers from the body. With lesions in the lower part of the brain stem, we usually find an alternating hemianalgesia, i.e. in the face on one side and in the body on the other side. In high lesions there is homonymous hemianalgesia.

Twenty-four hours later he appreciated pinprick in the upper extremities so that we cannot rely too much on the sensory findings. In patients who are in semicoma, sensory examinations cannot be depended upon too much; though we may infer therefrom we would not be willing to place too much emphasis on them.

The deep reflexes in the left extremities were more active than those in the right, which goes with the left motor weakness. The abdominal reflexes were unobtainable, which carries little weight in a man of 73, but it is of some significance in connection with the bilaterally extensor plantar responses. The patient had paralysis of the bladder.

When we find a combination of paralysis and sensory loss, we think first of a cortical lesion involving the motor and sensory pathways. In this patient we would exclude that possibility because he also had cerebellar findings and disordered eye movements. One may see paralysis of conjugate deviation in cortical lesions, but the severe disorder of the eye movements and the cerebellar signs would certainly indicate a lesion in the brain stem.

If we locate the lesion in this patient in the brain stem, his symptoms were due to involvement of the cranial nerve nuclei and of pathways in this region. Disturbance of the medial longitudinal fasciculus would probably explain the eye disorder, disturbance of the corticospinal tract, the complete paralysis on one side; and the involvement of ascending sensory pathways, descending sympathetic pathways, the cerebellar connections, and perhaps also the vestibular apparatus and supranuclear control of the tenth nerve would complete the anatomical picture.

The one finding that does not fit in well with the lesion in the brain stem is the initial convulsion. Lesions in the brain stem usually do not cause convulsive seizures. Our diagnosis would have to take this into consideration and we must find an explanation for both the lesion in the brain stem and the convulsion. It is difficult to do so. We shall delay for a minute consideration of the seizure and see if we can determine the etiology of the brain stem lesion.

In an individual with neurological symptoms of sudden onset one first thinks of a vascular lesion. The rule of thumb is that if symptoms are of sudden onset they are of vascular origin; of subacute onset, inflammatory or degenerative; and of slow onset, neoplastic origin. This man's signs were of sudden onset and should be of vascular origin. Support for this hypothesis is the suggestive history of a previous cerebral vascular lesion, he was of the age group where vascular lesions are common and there was evidence of vascular disease of the heart in the electrocardiogram.

If we conclude that the lesion was of vascular origin, was it of a thrombotic, embolic, or hemorrhagic nature? Hemorrhages into the brain stem are rather uncommon except as an accompaniment of sudden increased intracranial pressure such as may be seen with brain injury or terminal states of brain tumor. On this basis we would discard the diagnosis of hemorrhage. Embolism cannot be diagnosed clinically unless we find a focus from which an embolus may arise; I find no such source in our patient's record. We

therefore conclude that the lesion in this patient was thrombotic in nature. Having committed ourselves to a thrombotic lesion in the brain stem, what of differential diagnosis?

Neoplasms do occur in this region, they are more common in younger folk and it would be extremely rare for a brain stem glioma to have caused death so soon. Metastatic tumors may also occur in this region, but the same argument may be used against them; the course was too rapid. Abscess of the brain stem is extremely rare and I do not think we need to consider it. Another possibility is disorganization of the brain secondary to a severe cerebral lesion such as a large tumor or extensive cerebral trauma. There is nothing in the history to support this diagnosis and I believe it should be excluded.

If we do finally make our diagnosis vascular lesion in the brain stem, thrombotic in nature, what vessel was affected? The symptoms and signs were so diffuse that it is hard to enumerate individual nuclei and tracts and say that this lesion involved a single portion of the stem on one side. We cannot fit this clinical picture with the syndrome of the posterior inferior or any of the other cerebellar arteries and it certainly is not the picture of inefficient supply via the short vessels which perforate the brain stem. The patient had signs and symptoms pointing to the paramedian area and the lateral area and we cannot put our finger on a single small blood vessel, stoppage of which would give all these signs and symptoms.

What of the larger blood vessels that give rise to smaller ones? All of the arteries which supply the brain stem arise from the vertebrals or their continuation, the basilar, so we must consider whether thrombosis of the basilar artery fairly high in its course could be the cause of the patient's lesions. Could thrombosis of this vessel explain his convulsion? I believe it could. The occurrence of convulsions has been reported with thrombosis of basilar artery. What is the reason for the occurrence of such convulsions? The continuation of the basilar artery is the posterior cerebral arteries which supply the occipital lobe and a portion of the temporal and parietal regions. Sudden occlusion of the basilar artery could disturb the circulation of these parts of the cortex and give rise to a fit. If this happened in our patient why didn't he have visual field defects. He may have had a transient hemianopia which cleared up before he was examined but it is more likely that the nutrition of the occipital lobes was carried on by collateral circulation through the circle of Willis. Such collateral circulation however need not have prevented the convulsion which followed a transient disturbance of this area.

My impression of this case would be that the patient had a lesion of the basilar artery with destruction of fairly wide areas in the brain stem at the level of the pons and the midbrain. He perhaps might have had some softening in the occipital lobe or not, depending on whether the collateral circulation through the circle of Willis was adequate. This lesion was thrombotic in nature.

Dr. George L. Engel: Couldn't the convulsion be explained on the basis of cardiac standstill or fall in blood pressure at the time of the lesion? We have seen patients with basilar artery involvement who had syncope.

Dr. Merritt: I do not know. It has not been my experience that the blood pressure falls with lesions in the brain stem. I think you would have some difficulty explaining a convulsion by a lesion in the brain stem alone.

Dr. Milton Rosenbaum: This man had a fit and thereafter could not move his left side. Would Doctor Merritt consider the possibility that the paresis of the left side may have represented temporary weakness which sometimes follows a convulsion?

Dr. Merritt: It is my impression that the initial lesion was in the brain stem, but it is quite possible that this man had a cortical softening also.

Dr. I. Mark Scheinker: Would Doctor Merritt tell us whether he believes a cortical lesion in the cerebellum might produce the seizure?

Dr. Merritt: I do not believe it will produce a tonic-clonic seizure. In the so-called cerebellar fit the patient becomes comatose and goes into tonic spasm. It is my impression that this man had a tonic-clonic fit.

Dr. Aring: That was our impression too, though no one on the service saw the attack or heard it described adequately.

Dr. Joseph P. Evans: The clinical picture was predominantly one of unilateral dysfunc-

tion. Does that disturb you as far as basilar artery thrombosis is concerned?

Dr. Merritt: In the reported cases[1] the symptoms may be symmetrical, e.g. quadriplegia; or entirely unilateral.

*Discussion of Pathology*

Dr. I. Mark Scheinker: The visceral findings consisted of carcinoma of the prostate gland with multiple metastases to the liver and lungs. The kidney contained small foci of hemorrhagic infarction associated with tremendous congestion and stasis of the smaller veins.

On gross examination circumscribed focal areas of softening were found within the brain stem and cerebellum. Coronal section of the brain stem at the level of the substantia nigra and pons disclosed a linear area of softening (Figure 1), transecting the right half of the brain stem, involving the right cerebral peduncle and the substantia nigra of the right side and of the decussation of the rubrospinal and ventral tegmental tracts. The softening extended slightly beyond the midline toward the left.

Coronal sections through the cerebellar hemispheres revealed two areas of softening, almost symmetrically placed (Figure 2). That on the right (both were on the superior surface of the cerebellum) showed evidences of hemorrhagic infarction confined chiefly to the gray matter. The corresponding region of the left superior cerebellar surface disclosed a circumscribed area of grayish-brown discoloration, grossly resembling softening of recent origin. The remainder of the brain appeared grossly normal.

Microscopically, sections were examined from both cerebellar hemispheres and from the brain stem. Numerous small, and medium-sized veins were found to be occluded with thrombi only partly adherent to the vessel wall, and composed of strands of fibrin mixed with white and red blood cells; some of the thrombi contained a large mass of granular debris formed by broken-down white blood cells. The walls of the occluded veins appeared fairly well preserved except for slight loss of stainability of the cellular elements. Many veins and capillaries were tremendously distended by congestion and stasis. Numerous capillaries were surrounded by cuffs of extravasated red blood cells.

Changes in the nervous parenchyma, secondary to the vascular lesions, showed an early stage of hemorrhagic softening. There were numerous, small, petechial hemorrhages and tissue liquefaction and edema. In some areas the nerve cells showed signs of far advanced ischemia. The large majority of the lesions were characterized by complete disintegration of all tissue elements including the glia. No signs of proliferative glial reaction

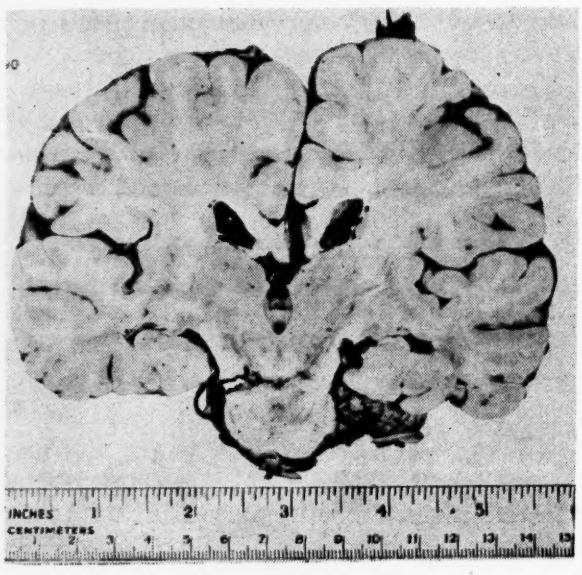


FIGURE 1

Coronal section of the brain transecting the third ventricle and substantia nigra. Note linear area of softening of the brain stem traversing the substantia nigra and cerebral peduncle on the right extending a bit beyond the midline to involve the mesial portion of the substantia nigra on the left.

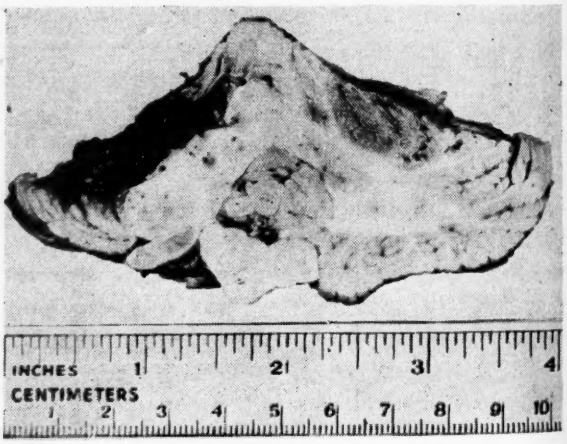


FIGURE 2

Coronal section of the cerebellum to show grossly symmetrical, bilateral areas of softening on the superior surfaces of the cerebellar hemispheres.

and no gitter cell formation could be disclosed. These parenchymal and vascular alterations have been described under the heading of vasothrombosis.[2]

Little is known about the pathogenesis of vasothrombosis. Study of numerous cases has brought forth the fact that thrombus formation may develop in healthy blood vessels, the walls of which are without any indication of a primary cause of thrombosis. Thus, it would appear that the pathogenic mechanism might be found within the blood stream. It is my impression that slowing down of the cerebral circulation for any cause but especially by cardiac failure ought to be considered as favoring the development of this pathologic condition.

*Clinical Diagnosis*

Softening, right midbrain on the basis of functional vascular disorder.

*Doctor Merritt's Diagnosis*

Thrombosis of the rostral portion of the basilar artery.

*Anatomical Diagnosis*

Softening, right midbrain, and bilaterally in the superior portion of the cerebellar hemispheres on the basis of vasothrombosis.

Carcinoma of the prostate gland with metastases to the liver and lungs.

Congestion of the kidneys.

Dr. Merritt: Could Doctor Scheinker explain why this disorder should be so symmetrical in both halves of the cerebellar hemispheres? Is it possible that the vessel changes you find in the softened area are secondary to that softening and not the cause of it? Bilateral cerebellar softening is not an uncommon finding after thrombosis of the basilar artery.

Dr. Scheinker: The symmetrical distribution of the lesions within the cerebellum is difficult to explain. Similar bilateral lesions have been observed in the temporal and occipital lobes in cases of post-traumatic vasoparalysis and in cerebral embolism.

In regard to the possibility that venous thrombosis may be a secondary reaction to cerebral softening; the latter we see with relative frequency and yet venous occlusion has in my experience never been observed as a sequel to it. In the case presented there were to be seen some thrombosed veins outside of the focal area of softening; they were found within relatively well preserved tissue and therefore could not be interpreted in terms of a secondary reaction.

The vessels of the circle of Willis were normal to gross examination with the single exception of a plaque of arteriosclerosis at the junction of the internal carotid and posterior communicating arteries on the right, which reduced the lumen of the latter vessel to an extremely narrow channel. Microscopically no signs of arteriosclerosis were seen in the arteries found in the sections taken from the cerebellum and brain stem.

Dr. Merritt: Is there any explanation for the fit?

Dr. Scheinker: In the clinical chart there is one casual remark that the patient had a tonic fit. If that were so, it would tally with your description of cerebellar fit.

Dr. Aring: It should be recalled that House Officers are addicted to medical "jargonese," and that such a casual notation cannot be relied upon. Nowhere in the record did I find the slightest description of the convulsion, and this is the extent of the information on this factor with which we had to work at the bedside.

Dr. Eugene B. Ferris: Was the examination adequate to rule out completely obstruction of an artery, either thrombotic or tumor? I don't think it is necessary to have severe arteriosclerosis to get a thrombotic infarct. The distribution of lesions in this case is, as Doctor Merritt has shown in the slides, consistent with basilar artery occlusion. It seems to me that venous disturbances and particularly stasis of blood in veins may result with hypoxia from any type of arterial occlusion, whether vasospastic or permanently occlusive. It is difficult for me to see why the primary lesion is limited to the particular distribution, if this is exclusively a venular lesion.

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# Comment on "A Near Fatality from Curare"

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In the recent communication[1] to this journal, describing a near fatality following curare, it would seem that Beard and Harris have given insufficient consideration to the possibility that their patient is suffering from myasthenia gravis. A female patient of 26 is described who complained of episodes of weakness and drawing about the lips, difficulty in swallowing and enunciation, and weakness in the upper extremities, occurring in paroxysms and alleviated by rest. The age, sex, presenting symptoms are characteristic for myasthenia gravis. It is not unusual for these patients to present in addition a reactive depression. The sequence of events following a comparatively small amount of curare would tend to confirm the diagnosis. For a patient of 120 pounds (54.4 Kg.) the ordinary curarizing dose would be 60 mg. of standardized curare. The patient received only 45 mg. of curare later proven to be of standardized potency, yet showed symptoms ordinarily associated with a massive overdose of curare. Further, the amount of prostigmin used (although the exact amount is not stated) would appear to be considerably greater than that required when an ordinary patient is excessively curarized. Usually one or two injections of one cc. of a 1:2000 solution of prostigmin is sufficient to overcome the effects of excessive curarization. The immediate response of the patient to insertion of an intratrachial airway is characteristic of that of one whose larynx is paralyzed by curare.

One is led to agree with the fourth explanation offered by Beard and Harris that "this patient was . . . . unduly sensitive to ordinary safe doses of curare and that her response to 45 milligrams of curare was an exaggerated reaction in keeping with the pharmacological effects of the drug when administered in large doses." However, this undue sensitivity to curare is used as a diagnostic test[2,3] for myasthenia gravis, although the dose of curare recommended for the diagnostic test is one twentieth to one fifth (5 to 15 mg.) of the average adult dose.

It should be remarked in passing, that the patient's convulsions were probably the result of anoxia, and are to be observed in cerebral anoxia of any type. Curare has a central depressing effect, although this is not ordinarily noticed since it is so overshadowed by its peripheral action.

The authors are to be congratulated for the energetic and successful treatment given the patient. However, it is usually safer to add atropine when large doses of prostigmin are given, especially to a patient with myasthenia gravis.

The case report is an important one. It demonstrates that even experienced and careful observers may not always succeed in establishing a diagnosis of myasthenia gravis, which may now seem obvious only as a result of what followed the administration of curare. The report should emphasize the importance of careful evaluation of the patient's physical as well as mental status before shock therapy is contemplated. It is especially important to be on guard for myasthenia gravis where curare is contemplated. If a positive diagnosis cannot be made, it would be worthwhile giving the patient the curare sensitivity test of Bennett and Cash. Further the report demonstrates the necessity of being prepared for all eventualities, including a paralyzed larynx, when administering curare. No drug is safe unless proper precautions are used. When properly administered[4] curare is a valuable adjunct in shock therapy.

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# Standard Treatment of Epilepsy*

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New medicines for treatment and modern opinions of the nature of seizures have altered greatly the outlook for the future of the epileptic person. Approximately one-half of the cases adequately treated with one or a combination of the anticonvulsant agents available today can be completely free of attacks so long as the treatment is administered regularly every day. An additional 25 per cent are markedly improved. The remaining 25 per cent require special attention whether they are at work, at play or in school.

This last group is rapidly becoming smaller and smaller as the medical, dietary, surgical and psychiatric treatments are more carefully integrated. Rehabilitation and resocialization of the epileptic person is decidedly helpful in reducing the number of seizures.

A thorough physical examination, neurological survey, psychiatric investigation and laboratory study determines the potential socio-economic value of the epileptic person. Seizures in a person otherwise physically and mentally normal are of slight or no hindrance to anything he might want to do.

Up to the first World War no one was aware that the incidence of epilepsy in the United States is as high as 0.5 per cent, that is, one epileptic in every 200 persons of the general population. Most authorities were amazed to know that there are as many cases of epilepsy as of diabetes or active tuberculosis. This estimated incidence of 600,000 to 700,000 in this country makes epilepsy one of the major medico-socio-economic problems that the medical profession is faced with today.

Sir Charles Locock[1] used bromides in the treatment of epilepsy on the hypothesis that a sedative would decrease the number of attacks. He was correct in this assumption. However, unpleasant symptoms were caused so frequently that many of the patients preferred having an attack rather than the discomfort of the medicine. This was the first real attempt to direct treatment of epilepsy to its principal seat, namely the nervous system. In the early part of the 20th century Hauptmann[2] substituted phenobarbital for bromides. Seizures were more effectively controlled and unpleasant side effects were less frequent and less severe.

Bromides, phenobarbital or a combination of these medicines were the nucleus of the treatment of epilepsy until about 1936. Merritt and Putnam doubted the value of sedation. They found that reduction in the number of seizures was out of proportion to the degree of sedation caused by bromides or phenobarbital. The barbiturates seemed to have a therapeutic agent which they chose to call "anticonvulsant" in contrast to the "sedative" effect.

A thorough search into the structural formulae of the medicines that reduce the number of seizures was made. Two common elements were found to be present and this suggested that the anticonvulsant property of a drug depends upon the relative concentration of these elements. Obviously it was impractical to clinically test in the human all the compounds known to science that contained these two elements. After the methods of Krosnogoesky and Spiegel a simple test was devised by Putnam and Merritt whereby the anticonvulsive property of a medicine could be determined accurately. A known quantity of an electric current is passed through the brain of a cat by electrodes suitably placed in the animal's mouth and on the intact scalp. The amount of electric current required to cause the animal to convulse is a constant factor of the test from day to day. It is increased consistently by the medicines that are known to reduce the number of seizures and in doses that cause no toxic symptoms in the experimental animal.

It was demonstrated by Putnam and Mer-

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ritt[3] that electric currents of less than 10-20 milliamperes never induced a convulsion in the animal but those of greater strength were invariably followed by a generalized seizure. This property of nervous tissue is now known as the "convulsive threshold". Just what determines the inherent threshold of nervous tissue to respond to a stimulus is not known nor is the mechanism by which anticonvulsants raise the convulsive threshold known to us.

One of the first compounds tested experimentally raised the convulsive threshold so effectively that in some animals a convulsion could not be induced with an electric current as high as 50 milliamperes. It was 5.5 diphenylhydantoin. The sodium salt of this compound is known to physicians as phenytoin sodium and to the public as dilantin. Its anticonvulsant effect experimentally and clinically is greater than phenobarbital and bromides as well as other compounds structually similar to it. The results of the experimental survey of dilantin sodium has been confirmed in the human by elimination or reduction of the number of seizures in the epileptic person and prevention of induced seizures in shock therapy for mental illness.

On the basis of the anticonvulsant property of certain medicines to raise the convulsive threshold, an attempt is being made to standardize the drug therapy of epilepsy. The use of these medicines imply that fundamentally they act in one of two ways. One type of action is that the drug depresses the function of nervous tissue. The second is the medicine, or some of its breakdown products, replaces a chemical substance that is lacking in the epileptic person and raises the convulsive threshold to or above its normal level. Either of these hypotheses are possible methods of action of the anticonvulsant medicines. The second lends itself for investigative purposes more readily than the first one. Moreover, the second one suggests that epilepsy is a disease or symptom complex caused by an inherent absence or acquired loss of specific chemical substances which are replaced by medicines known to raise the convulsive threshold.

Unfortunately anticonvulsant medicines have, in addition to the specific chemical substance required to raise the convulsive threshold, other properties that cause unpleasant symptoms. The undesirable side effects of many medicines prevent their use in the treatment of human illness. Anticonvulsant medicines are no exception. Furthermore, sensitivity to medicines varies from person to person. The successful treatment of epilepsy consequently depends on the physician's ingenuity and experience in the art of the practice of medicine reinforced by a dogged tenacity to succeed on the part of the patient. Both must be willing to accept failures and enjoy success in order to find the dosage of one or a combination of anticonvulsant medicine suitable to relieve the circumstances presented by the epileptic person.

A standard treatment for epilepsy may be defined as that amount of anticonvulsant medicine that will control the seizure and yet cause no unpleasant reactions. To reach this goal, medicines must be used systematically to prevent unnecessary confusion and repetitions of ineffective methods.

The nucleus of treatment is phenytoin sodium (dilantin). The initial dose is 0.1 gram administered just before the patient retires for the night. The initial dose is small because toxic symptoms, namely ataxia, drowsiness, blurring of vision, morbilliform rash, occasionally fever and infrequently gastric distress, are too commonly caused by starting medication with doses as large as 0.3 to 0.4 grams. The small dose of dilantin is continued for one week. An additional capsule of 0.1 gram is added each successive week until 0.4 to 0.5 grams is being administered. Increase in the daily dosage is stopped only in case toxic symptoms appear in which case the dosage may then be decreased by 0.1 gram. The medicine need not be discontinued completely to alleviate unpleasant symptoms, in fact, it is a dangerous practice because status epilepticus may be induced.

Recurrence of seizures usually indicates that an inadequate amount of anticonvulsant medicine is given. There are no clinical tests that may be utilized as an index or estimation of the amount of phenytoin sodium absorbed and assimilated. Overdosage symptoms are the only positive criteria that can be used to prevent inadequate therapy. After the maximum dose of phenytoin sodium has been

determined and seizures recur phenobarbital is administered in increasing doses. If the amount required to control seizures also causes unpleasant symptoms, the medicines used are unsuitable for continuance of treatment. The alternative is to substitute mebaral for phenobarbital, continuing the maximum dosage of dilantin. The same procedure for determining the dosage level is utilized as with the other medicines mentioned above.

In case seizures recur after a reasonable trial of the subtoxic dosage, phenytoin sodium is replaced in the treatment by phenatoin (Methyl phenxyl ethyl hydantoin). It is administered similarly to the method described for phenytoin sodium. Unpleasant symptoms caused by phenatoin are identical to phenytoin sodium except that it does not cause hypertrophic gingivitis.[4,5] If present, gingivitis is lessened during treatment with phenatoin even without the other forms of therapy designed specifically for it, namely, massage and surgical removal of the excess tissue.

The medicinal therapy described above is most effective in reducing the number of grand mal attacks. Psychomotor seizures and psychic equivalents are equally reduced in frequency. Petit mal attacks are affected least by these medicines but there are others which make the lives of these unfortunate persons much more pleasant.

Glutamic acid[6,7] administered in doses of 8 to 16 grams daily reduces the number of petit mal attacks in about one-fourth to one-third of the cases. It is rather cumbersome to administer glutamic acid as a medicine because the dosage must be divided into 4 to 8 tablets given four times a day. The tablets are each one-half gram. The advantages are that the patient is more alert mentally and physically, and the toxic symptoms are minimal—usually limited to gastric distress.

A new medicine which is now available to everyone is 3,5,5—trimethylaxazolidin —2-4 dione more commonly known as tridione. Lennox[8,9] recently reported complete relief of symptoms or 75 per cent improvement in all but seven cases out of 40 that had been treated adequately. It is the first drug that has consistently reduced the number of petit mal attacks. The toxic symptoms are not too severe to enable a number of those treated with tridione to continue with the medicine and remain free of symptoms.

The unpleasant symptoms most commonly mentioned are visual in nature. As one person stated, "The leaves of the tree are covered with milk or snow." There is some photophobia. With larger doses drowsiness, lethargy and headache are noticed. The dosage of tridione depends on the response of the patient. It varies from 1 to 2 grams a day given in divided doses of 0.3 grams. The drug is supplied in 0.3 gram capsules.

Medicinal therapy is adequate only when each of the medicines mentioned have been administered alone or in combination in amounts large enough to control seizures and yet cause no unpleasant symptoms. There are some cases that do not respond to medicines regardless of the care with which they are administered. Dietary, surgical and psychiatric treatment are intergrated with medicines to obtain the best results.

The only diet that has been proven to be effective in reduction of the number of seizures is the ketogenic diet or "high fat diet".[10] It has been found most useful in the treatment of petit mal seizures in children. Few investigators have enthusiastically utilized this method of treatment since it is a dietary and psychological burden to a child or adult.

Surgical treatment has been most thoroughly studied by Penfield.[11] There are cases that improve after surgical excision of a scar or "epileptogenic focus," but a very small percentage of patients fit into this category. This type of therapy is delegated to those who are especially trained in detecting the relative excitability of different areas of the cortex and cerebral localization.

The psychiatric management of the epileptic person is not particularly different from the care of other patients with chronic illnesses with the exception that a mass of misinformation has been accumulated in the public mind regarding the causes and sequellae of epilepsy. The average patient and his relatives must be thoroughly re-educated regarding these aspects. Endless questions must be answered and a simple honest explanation of what we do and do not know about seizures must be made. The patient is given detailed instructions regarding medication and is encouraged to return at frequent

intervals for readjustment of the dosage until such a time as his seizures are controlled.

In the epileptic child the responsibility for taking medication is turned over to him as soon as he is old enough to assume it. Unnecessary restrictions should be removed and the child should be treated as a normal individual insofar as it is possible. Many epileptic children are more seriously handicapped by a superimposed personality illness induced by overprotective parents than they are by their seizures.[12]

Psychoneuroses and psychoses appear in epileptic patients in about the same proportion as they do in the general population. These illnesses occurring in epileptics respond to psychotherapy or the special therapies such as insulin shock, electro-shock, or prefrontal lobotomy as well as they do in patients without epilepsy. Too frequently the psychiatric symptoms are attributed to the epilepsy. Epileptic equivalent states or the post-seizure confusional states are often incorrectly interpreted as behavior disorders. Mental retardation and toxic delirious reactions may be the result of the toxic effects of the anticonvulsive medication rather than evidence of deterioration in an epileptic. In these cases remarkable improvement is noted when the proper medication and dosage is substituted.

### *Summary and Conclusions*

The successful treatment of epilepsy depends on the following measures:

1. Medicine must be taken regularly every day regardless of the frequency of seizures.

2. The hydantoin series of drugs may be administered in one dose as effectively as in divided doses.

3. Medicines should be changed slowly; for example, one to three months is required to change from 0.3 grams of phenobarbital to a similar or larger dose of dilantin.

4. Toxic symptoms are alleviated by reduction of the dosage of phenytoin sodium. Usually 0.1 grams is an adequate amount to decrease the dose so that the patient may be comfortable and yet have no attacks. Rarely is it necessary to reduce the dosage 50 per cent or more.

5. The optimum dosage is the amount of medicine or combination of drugs that reduces the number of seizures and yet causes no unpleasant symptoms.

6. The patient must be encouraged to understand, accept and live with his illness.

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